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Damage of the Nervous System in Chronic Kidney Diseases: Clinical-Pathogenetic Mechanisms

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Annotation: Under physiological conditions, the as ab system and the kidneys communicate with each other to maintain normal body homeostasis. However, pathological conditions, as seen in hypertension, disrupt this interaction , and renal damage can lead to impairment of the renorenal reflex and sodium handling.

Keywords: kidney, chronic kidney disease, stroke, hemodialysis, vagus nerve , splenic nerve, dementia.

In acute kidney injury (AKI) and chronic kidney disease (CKD) can have detrimental effects on the central nervous system. CKD is an independent risk factor for cerebrovascular disease and cognitive impairment, and multiple factors, including uremic toxins and phosphate retention, are CKD-specific factors responsible for structural and functional brain changes in CKD patients. offered as However, more research is needed to determine the exact pathogenesis. Chronic kidney disease (CKD) is a significant health burden worldwide, with an overall prevalence of 13.1% in the United States (1) . Neurological disorders, including cerebrovascular disease and cognitive impairment, are common in patients with CKD. The annual incidence of stroke is 15.1 % in hemodialysis patients and 9.6% in CKD patients, while the annual stroke rate in non-CKD patients is 2.6% (American Qo United States Renal Data System 2006. Epidemiological studies have shown that AKI is associated with subsequent risk of stroke and dementia. On the other hand, recent animal studies have shown that renal nerve i contributes to renal inflammation and fibrosis, while activation of the cholinergic pathway involving the vagus nerve and splenic immune cells has a significant renoprotective effect , elucidating the communication mechanisms between the nervous system and the kidneys will not only allow the development of new strategies to improve the neurological conditions associated with kidney disease, but also the development of safe and effective clinical interventions for kidney disease using the neural and neuroimmune control of the kidney. injury and illness.

and serious medical problems worldwide. This disease not only destroys the functioning of the kidneys, but also causes great damage to other organs and systems, especially the nervous system. Toxic substances and metabolic disorders accumulated in the body due to SBK cause damage to the nervous system, which reduces the quality of life of patients and complicates the disease process. This article is dedicated to the in-depth study of damage to the nervous system and its clinical-pathogenetic mechanisms in chronic kidney diseases.

The nervous system and kidneys interact to maintain normal body homeostasis. However, pathological conditions such as hypertension and kidney damage can disrupt this interaction, which subsequently leads to the loss of normal homeostasis.

Recent studies have shown that the nervous system can directly or indirectly affect the course of AKI and CKD through the immune system (neuroimmunomodulation). In this review, we discuss interactions between the nervous system and the kidney, focusing on possible underlying mechanisms contributing to neurological disease in patients with CKD and AKI, and the neuro/neuroimmune control of kidney disease. , which is a new and promising therapeutic target. patients with kidney disease.

The nervous system and the kidney communicate in different ways to maintain a normal physiological state. For example, neuroendocrine/renal interactions are responsible for regulation of blood

osmolality by vasopressin (4 - 6). Systemic changes in osmolarity are detected by osmoreceptors expressed in certain regions of the central nervous system. Neuronal activity in these regions regulates vasopressin secretion from the hypothalamus/posterior pituitary gland and also stimulates or inhibits thirst and sodium appetite. Circulating vasopressin acts on vasopressin 2 receptors expressed in the renal collecting duct, which increases the number of aquaporin-2 channels in the apical membrane and enhances renal water reabsorption.

Another example is the renal sensory afferent and sympathetic efferent nerves, which work together with the kidney to maintain sodium balance (3). Renal sensory nerves, which innervate mainly the pelvic wall and, to a lesser extent, the renal vessels and parenchyma (4), are excited by increased pelvic pressure and pelvic wall stretching (10, 11). In contrast, sympathetic nerves innervate the entire renal vasculature, with the densest innervation to the afferent arterioles; tubules are also innervated by sympathetic nerves (12). Thus, norepinephrine released from sympathetic nerve terminals can directly affect renal vessels and tubular epithelial cells. Increased efferent renal sympathetic nerve activity (ERSNA) increases renin secretion (juxtaglomerular granule cells), decreases urinary sodium excretion (tubular epithelial cells) and renal blood flow (vascular smooth muscle cells). In normal rats, unilateral renal denervation (ablation of both sensory afferent and sympathetic efferent nerves) increases ipsilateral urinary sodium excretion (due to sympathetic ablation), and increased contralateral ERSNA decreases contralateral urinary sodium excretion (13). Stimulation of renal sensory afferent nerves decreased contralateral ERSNA with an increase in contralateral urinary sodium excretion (2). These findings support the existence of an inhibitory renorenal reflex—an increase in afferent renal nerve activity (ARNA) suppresses ERSNA. Several studies have shown that increased ERSNA increases ARNA (10), suggesting a negative feedback system between ERSNA and ARNA.

Among the main causes of chronic kidney diseases are diabetic nephropathy, hypertensive nephropathy, glomerulonephritis and other kidney diseases. During these diseases , the kidneys gradually lose their function, as a result of which changes are observed in the blood-vascular system, heart and other organs . consists of:

- Fatigue and general weakness;
- Dehydration or swelling;
- Increased blood pressure;
- Violation of water and electrolyte balance;
- Anemia.

In addition, due to the effects of SBK on the nervous system, neurological symptoms may occur, which are more pronounced in the complicated form of SBK.(11)

Harmful effect on the nervous system - During chronic kidney disease, the nervous system also suffers a lot. This lesions cause varying degrees of neurological symptoms and complications, such as:

- Peripheral neuropathy is caused by damage to the peripheral nerves and manifests itself mainly in the form of numbness, pain, weakness and movement problems in the limbs. Nerve damage from toxic substances that accumulate due to SBK is the main cause of peripheral neuropathy.
- Brain dysfunction (encephalopathy) - changes in brain activity occur as a result of kidney failure not being able to excrete toxins from the body. Patients may experience blackouts, cognitive impairment, or even coma.
- Myelopathy is another type of damage to the nervous system associated with kidney failure.

There is a complex pathogenetic relationship between the nervous system and the kidneys. Damage to the nervous system due to SBK occurs through different mechanisms:

- Metabolic changes : When kidney function is impaired, toxins (urea, creatinine and other substances) accumulate in the body, which has a toxic effect on nerve cells. At the same time, electrolyte and acid-base imbalances irritate nerves and start the process of degeneration in them.
- Oxidative stress: Kidney failure results in an increase in free radicals, which expose cells, including nerve cells, to oxidative stress and damage their structure and function.
- Hyperparathyroidism : Secondary hyperparathyroidism develops as a result of impaired phosphorus and calcium metabolism in renal failure . This directly affects the nervous system and causes changes in nerve fibers and their membranes.
- Immune system and inflammatory processes : Impaired immune response and overproduction of inflammatory mediators in SBK can lead to nerve tissue damage. These inflammatory processes also damage the brain and peripheral nervous system.

In recent years, many studies have been conducted on damage to the nervous system in chronic kidney disease. Studies show that prescribed dialysis or a kidney transplant can improve nervous system function in some stages of treatment, but complete recovery is not always observed in advanced stages of the disease. Studies have also shown that peripheral neuropathy occurs at a higher rate among patients with SBK, and that diabetes is a major risk factor in this condition.(14)

Summary. The effects on the nervous system of CKD patients are complex and multifaceted, further increasing the severity of the disease. Damage to the nervous system and its mechanisms should be considered in the treatment of the disease. Early diagnosis and treatment, including control of blood toxin levels, metabolic rebalancing, and dialysis or kidney transplantation, are essential to prevent and treat these lesions.

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